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ABSTRACT

The paper reviews a theory advanced by Satz and Sparrow (1970) which purports to explain the nature and cause of specific developmental dyslexia, and evaluates several developmental hypotheses which are generated bythe theory. The theory postulates that developmental dyslexia is not a unitary syndrome but rather reflects a lag in the maturation of the brain (left hemisphere) which delays differentially skills which are in primary ascendancy at different chronological ages. It is concluded that, instead of determining whether the primary handicap in dyslexic children is perceptual, linguistic, or both, findings suggest that the nature of the handicap will vary largely as a function of chronological age. (Author/KW)

Developmental Dyslexia: An Evaluation of a Theory[1]

Paul Satz and Gary K. Van Nostrand[2]

INTRODUCTION

In recent years considerable research has been addressed to the problem of specific reading disability. This research has focused on a broad range of factors comprising definition of terms, nature of the disorder, incidence rates, etiology, treatment and prognosis. Despite progress in attempts to understand this disorder, many questions and considerable controversy still exist.

Before discussing the problems it should be stated that there is now general agreement that there are multiple determinants of reading disability, including sociopsychologic (deficiencies in teaching, cognitive stimulation and motivation), psychophysiologic (defects in sensory, intellectual and brain function) and genetic variables (hereditary transmission from parents to offspring).

Eisenberg (1966) has shown that the incidence of reading disability increases inversely and dramatically with socio-economic status. Cultural deprivation, crowded schools and poor motivation definitely contribute to deficiencies in reading and language facility (Bloom, 1964). Brain-injury, and more typically, low intellectual ability have also been associated with reading handicap (Ingram, 1970). One might argue that the latter factors (brain injury and low IQ) are more frequently observed in lower socio-economic families (particularly where malnutrition and poor health care prevail during early development) which would suggest that cultural deprivation might constitute a primary cause of childhood reading disability.

Unfortunately, the matter is not that simple. First, there is increasing evidence that many reading disabled children show a familial pattern in which one or both parents were handicapped readers as children, regardless of social class (Hallgren, 1950; Ingram, 1970; Owens, Adams & Forrest, 1968; Silver,

1971). Second, it has been shown that some disabled readers with a positive family history, who were born prematurely or with birth complications, have siblings without birth complications with similar learning disability (Silver, 1971). Third, it is recognized that there are many children with reading disability who have average or above average intelligence, educational and cultural opportunity and no evidence of structural brain injury, who nevertheless are delayed in reading and writing skills (Money, 1962; Myklebust, 1968; Critchley, 1968; Eisenberg, 1966; Satz & Sparrow, 1970). These children, moreover, do not always reveal a positive family history of reading disability (Silver, 1971). Fourth, regardless of the assumed "cause" of the disorder, investigators have uniformly reported a significantly higher incidence of males (approximately 6:1) in children with specific reading disability (Eisenberg, 1966; Ingram, 1970; Satz & Sparrow, 1970).

Although the evidence points to multiple determinants of reading disability, investigators have long puzzled over that subgroup of children who fail in reading and writing despite the existence of cultural and educational opportunity, at least average intelligence and freedom from gross sensory or neurological handicap. In fact, investigators have long implicitly recognized a bimodal distribution of reading disability, at least on superficial grounds, which attempts to explain this disorder in the absence of socio-cultural and neurological deficiency. Such studies have employed a number of different terms which nevertheless seem to refer to this special subgroup of disabled readers. They are as follows: congenital word blindness (Morgan, 1896); primary reading retardation (Rabinovitch, Drew, de Jong, Ingram & Withey, 1954); specific dyslexia (de Hirsch, 1968); strephosymbolia (Orton, 1928); educationally handicapped (Owens, Adams & Forrest, 1968); and psychoneurological learning disability (Myklebust, 1968).

The present chapter is addressed to this latter group of children who,

as a subgroup of the larger population of retarded readers, has recently been classified as cases of specific developmental dyslexia (Waites, 1968). The purpose of this chapter is twofold: (1) to review a theory recently advanced by Satz and Sparrow (1970) which purports to explain the nature and cause of this disorder and (2) to evaluate a number of developmental hypotheses which are generated by the theory.

I. THEORY

The theory postulates that developmental dyslexia is not a unitary syndrome but rather reflects a lag in the maturation of the brain (left hemisphere) which delays differentially those skills which are in primary ascendency at different chronological ages. Consequently, those skills which during childhood develop ontogenetically earlier (e.g., perceptual-spatial and cross-modal integration) are more likely to be observed in younger children who are maturationally delayed. Conversely, those skills which during childhood have a later or slower rate of development (e.g., language and formal operations) are more likely to be observed in older children who are delayed maturationally.[3]

Briefly, the theory is compatible with those developmental positions which postulate that the child goes through consecutive stages of thought during development, each of which incorporates the processes of the preceding stage into a more complex and hierarchically integrated form of adaptation (Hunt, 1961; Piaget, 1926; Bruner, 1968).

Satz and Sparrow (1970) state that this evolving process of development, characterized by different stages of thought, is facilitated by experience and by increased maturation and differentiation of the CNS. Further, it is assumed that this maturation process, in normal children, is largely a

function of chronological age (de Hirsch, Jansky & Langford, 1966; Gesell, 1945; Piaget, 1952).[4] A maturational lag, therefore, is defined as slow or delayed development of those brain areas (left hemisphere) which facilitate the acquisition of developmental skills which are fundamentally age-linked. This formulation therefore postulates that even in cases of delayed CNS maturation the child would continue to develop, albeit at a slower rate, and that his observed pattern of difficulties should change with increased age and new demands for skills which develop ontogenetically later. Thus, one would expect differential patterns of behavior, within disabled readers, as a function of age. It should follow, therefore, that the error patterns observed in dyslexic children should resemble the behavioral patterns of chronologically younger normal (P.4) children who have not yet acquired mastery of later developing skills. Construed in this light the behavioral performance of a disabled reader is explained as a lag or delay in acquisition rather than as an impairment or loss of function. This formulation therefore attempts to conceptualize the behavior within the context of a developmental rather than a disease model.

A critical postulate in the Satz and Sparrow (1970) theory is that the dyslexic child is handicapped on a number of developmental skills which are not directly or seemingly related to the reading process. This phenomenon has largely been unexplained by investigators who have examined specific correlative defects (e.g., right-left confusion, perceptual difficulty or depressed Verbal IQ) in these children (Belmont & Birch, 1966; Harris, 1957; Ackerman, Peters and Dykman, 1971). More recently, this phenomenon has been ignored or dismissed as irrelevant by investigators who have focused on direct operant intervention of the reading process (see Wolking, Chapter). Although there are obvious merits in attempts to shape the rate of oral and silent reading, using appropriate reinforcers, these methods

have not provided a theoretical explanation of the reading disorder nor of its antecedents (causal or otherwise). One might still ask why these children have long been reported to have difficulty in one or more of the following skills: right-left discrimination, finger sequencing and identification, writing and calculation ability, verbal intelligence, perceptual discrimination, perceptual-motor integration, auditory-visual integration, word fluency and the like (see reviews by Belmont & Birch, 1966; Ingram, 1970; Satz & Sparrow, 1970).

Explanation 1. One possible answer to the above question is that each of the preceding developmental skills may be differentially crucial to reading performance depending upon the level or age of the child. Gibson (1968) has already distinguished three sequential phases in the process of learning to read: (1) learning to differentiate graphic symbols, (2) learning to decode letters to sounds (i.e., mapping letters into sounds) and (3) learning to utilize higher-order units of linguistic structure. This analysis of the reading process is quite similar to Luria's (1966) analysis which distinguishes the following sequential stages: (1) perception of letters; (2) analysis of their conventional phonetic value and (3) complex fusion of phonetic letters into words. Each author recognizes an orderly and developmental sequence in which the early phases of reading are characterized by processes of perceptual discrimination and analysis. In this early phase the child must discriminate the distinctive features of letters (e.g., break vs. close, line vs. curve, rotation and reversal) before he can proceed to later phases which require more complex phonetic and linguistic analysis. This schema is, in fact, quite similar to Bruner's (1968) notion of cognitive development in which the early stages of iconic representation of percepts and images precede and influence later stages of symbolic and linguistic representation.

If the child, in the early phases, has difficulty in discriminating the essential units of form and orientation of letter stimuli, then he is bound to extract irrelevant information before he proceeds to the following and hierarchically more complex levels of phonetic analysis and fusion of phonetic letters into words. Unfortunately, the law of object constancy is not applicable to the perception of linguistic material (Money, 1962). An object such as a chair is perceived correctly by a child regardless of the spatial position of the object or perceiver. On the other hand, the perception of letter or word stimuli are instrinsically dependent upon their position and orientation in space (e.g., p vs. b, M vs. W, was vs. saw). It has already been shown that normal pre-school children (kindergarten) have difficulty in correctly matching the correct spatial orientation of even nonlinguistic designs to the target stimulus; however, this visuo-spatial skill improves as the child matures and it is usually asymptotic by seven years of age or when formal reading instruction is begun (Ingram, 1970; Small, 1968; Wechsler & Hagin, 1964). In fact, Wechsler and Hagin (1964) showed that performance on such visual-spatial tasks is correlated with both reading readiness and progress in beginning reading.

Explanation 2. A second answer to the preceding question is suggested when comparison is made between the performance of disabled readers, and adults who have sustained acquired lesions of the left cerebral hemisphere. Damage to the left or dominant speech hemisphere in adults has long been shown to produce impairment in one or all of the skills already observed in dyslexic children (Satz & Sparrow, 1970). This impairment, while quantitatively more severe in adults, is nevertheless similar to dyslexic children for whom no evidence of structural brain disease is apparent.

Maturation Lag Hypothesis (clue to mechanism). The explanation advanced to account for this similarity in performance between groups

contrasting in age and etiology was first suggested by Money (1966). He stated that ". . .The great majority of reading disability cases will be classifiable not on the basis of brain pathology, but simply as representative of a lag in the functional development of the brain and nervous system that subserves the learning of reading." (1966, p. 35). Satz and Sparrow (1970) extended this view by conceptualizing the behavioral signs within the framework of left hemisphere integration. They hypothesized that whereas damage to the left hemisphere in adults may produce temporary loss of a function, a delay in the maturation of the left hemisphere in children may retard the acquisition rather than the loss of the same function. This formulation thus incorporates both of the preceding explanations (1 & 2) in order to account for the variable pattern of performance difficulty in disabled readers. The first explanation is that many of the correlative difficulties are differentially related to the reading process depending upon the age or reading level of the child; the second explanation is that delays in the maturation of the brain, particularly the left hemisphere, retard the acquisition of those developmental skills which are temporarily related to reading.

If one postulates a causal relationship between level of brain maturation and behavior (performance acquisition), then one could begin to conceptualize the nature of the disorder in dyslexia within the context of developmental psychology. Although the neural mechanisms underlying CNS maturation are not, at present, subject to direct observation, the use of this formulation as a hypothetical construct does generate a number of developmental hypotheses (previously discussed) which are testable.

Sequential Stages in Development

Supporting Data: Behavioral and Neurological.

Before proceeding to a

review of the evidence pertaining to the above hypotheses (Satz & Sparrow, 1970), a brief discussion should be given to the concept of sequential and hierarchical stages of development in psychology and neurology.

Psychological. It has long been known that perceptual-spatial and perceptual-motor skills undergo primary development between the ages of five and nine (Elkind, Koegler & Go, 1962; Hunt, 1961; Piaget, 1926). Moreover, it has been shown that there is a hierarchical development within the process of perception with lower level recognition-discrimination skills preceding the development of more complex visual-motor skills (Birch & Lefford, 1964; Small, 1968; Tessler, 1971). Piaget (Hunt, 1961) has long advocated an orderly sequence in the structural development of intelligence in children with sensori-motor and pre-conceptual stages preceding, and influencing, the development of language and formal operations in later stages (ages 9-12).

Bruner (1968) has advanced a similar position suggesting that the child utilizes three different systems in cognitive development. He constructs models of his world through action (enactive representation), through imagery (iconic representation) and through language (symbolic representation). "Their appearance in the life of the child is in that order, each depending upon the previous one for its development, yet all of them remaining more or less intact throughout life" (p. 478). According to Bruner, the transition to symbolic representation marks the final and most important stage in cognitive development. This stage frees the child from dependence upon the concrete and immediate aspects of perceptual representation and, through language internalization, facilitates a so-called second-signal system (Luria, 1966) in which experience can be both represented and transformed.

Longitudinal studies on the growth of primary mental abilities also

reveal a sequential and differential rate of development (Bloom, 1964; Thurstone, 1955). Thurstone fitted Gompertz equations to longitudinal and cross-sectional data on the growth of special mental abilities from ages one to 19. The results are presented in Figure 1 and reveal a marked differential growth rate for the selected mental abilities. Perceptual and

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Insert Figure 1 about here

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spatial abilities reveal an earlier ontogenetic development whereas verbal abilities reveal a later and slower development with age. In fact, if one uses 80 percent of the adult performance as one index of comparison, it is apparent that the Perceptual Speed Factor reaches asymptote (Age 12) almost eight years before Word Fluency reaches asymptote (Age 20).

Neurological. Studies of normal and brain-injured children indicate that the brain undergoes extensive maturation during the first decade of life. This process of maturation is characterized by rapid growth during the first two years, marked, at this time, by the onset of speech, and that the rate slows down and reaches an asymptote around puberty--"at just about the same time that trauma to the left hemisphere begins to have permanent consequences" on language facility (Lenneberg, 1967, p. 167). There is evidence that during this period of rapid growth in brain maturation, language and speech become progressively and irreversibly lateralized to the left cerebral hemisphere. This period is proximal in time to the acquisition of conceptual or formal operations as defined by developmental psychologists (Hunt, 1961; Piaget, 1926).

Anatomical studies of the cerebral cortex also show a sequential and hierarchical development as the brain undergoes maturation. According to Geschwind (1968, p. 183): "The early myelinating zones include all of

the classic motor and sensory zones, i.e., the classical motor cortex (area 4) and the primary somesthetic, visual and auditory cortices." These early-myelinating or "primordial" zones have the most efferent and afferent connections with subcortical structures and the fewest long connections with other cortical areas. By contrast, those zones which myelinate latest (ages 9-12), the "terminal" zones (i.e., angular gyrus), have prominent intercortical connections which are necessary in the mediation of more complex language skills.

More recently, Semmes (1968) attempted to expand this concept of sequential stages in neural development to account for the phenomenon of hemispheric lateralization of speech. She proposed that the later development of speech specialization on the left might stem from a basic difference in sensorimotor organization which has already been specialized within the left hemisphere. This formulation therefore accounts for differences in the hemispheric organization of a complex function (e.g., speech/language) as an outgrowth or synthesis of elementary sensorimotor functions (e.g., "primordial zones") whose neural organization already favors specialization on the left. According to Semmes (p. 11): ". . .focal representation of elementary functions in the left hemisphere favors integration of similar units and consequently specialization for behaviors which demand fine sensorimotor control, such as manual skills and speech."

In a related vein, Lenneberg (1967) has suggested that the mechanisms of brain maturation constitute prerequisites and limiting factors for language and conceptual development. His position is that the attainment of brain maturation is correlated with behavioral achievements or "horizons" as depicted in Figure 2.

Insert Figure 2 about here

This figure describes the relationship between the level of brain maturation (composite estimate of various parameters) and behavioral achievements at different ages for two different populations. Comparison of the growth curves for the normal and retarded children show that the relative distances among the various milestones become greater with advancing age. For example, it can be seen that whereas normal children begin to join words together about 15 months after sitting up, the spacing between milestones may be as long as 24 months in retarded children. Inspection of this figure also shows that the spacing becomes even greater between joining words together and general language establishment in retarded children (five years) vs. normal children (two years). In other words, if the normal maturation function is slowed down, the developmental milestones are acquired later, and according to Lenneberg (p. 170), "the spacing between the milestones becomes more prolonged without altering the order or sequence. This is precisely what is found in generally retarded children. Their earliest milestones seem delayed by just a few months, but the delay is increased with advancing age, and the lag behind the norm becomes worse and worse even though the retarding disease may be stationary and maturation is progressing steadily but slowly."

The preceding formulation is quite compatible with the maturation lag theory advanced by Satz and Sparrow (1970) to account for developmental dyslexia.[5] Lenneberg (1967) not only proposes a relationship between level of brain maturation and behavior, but hypothesizes that a lag in the maturation of the brain will differentially delay the behavioral attainments of early vs. late-appearing milestones. Of particular relevance is Lenneberg's position that during childhood, the retarded child continues to acquire new behavioral horizons, although at a slower rate. He eventually succeeds in sitting up and he eventually succeeds in joining words together, although

the spacing between milestones is greater than in normal children. The point is that during childhood the retarded child reveals a delay rather than loss of ability to acquire many of the basic developmental milestones. More ominous is the notion that the spacing between horizons becomes greater as the retarded child becomes older. An attempt will be made, in the following section, to account for this phenomenon in the context of developmental dyslexia.

II. TEST OF THEORY: Developmental Hypotheses.

The preceding section reviewed the general postulates of the theory which described the nature of the disorder and the neural mechanism presumed to underlie the disorder (i.e., lag in maturation of left hemisphere). Evidence was also presented from the fields of developmental psychology and neurology which was felt to be compatible with the notion that (1) maturation of the cerebral cortex is correlated with behavioral attainment, (2) maturation of the brain follows an orderly and sequential process with elementary zones preceding the organization of later developing or terminal zones, (3) development of intelligence similarly follows an orderly and sequential process with each stage incorporating the processes of the preceding stage into a more complex and integrative operation and (4) that lags in the maturation of the brain will correspondingly retard the rate at which the behavioral acquisitions are obtained, without altering the sequence.

The following section is addressed to the hypotheses generated by the theory and an evaluation of the evidence pertaining to the hypotheses.

Hypotheses. The theory predicts that those skills which during childhood develop ontogenetically earlier (e.g., perceptual-spatial and cross-modal integration) are more likely to be observed in younger children who are maturationally delayed. Conversely, those skills which during childhood have a later or slower rate of development (e.g., language

and formal operations) are more likely to be observed in older children who are delayed maturationally. Implicit to these statements is the assumption that those basic skills (e.g., sensorimotor) which precede the development of perceptual-motor integration are not likely to be observed in younger dyslexic children because these "horizons" are generally established by ages 4-6 or before reading disability can be diagnosed. Thus, a child who is maturationally delayed at these ages (4-6) may show incomplete manual laterality which could forecast later reading problems. However, when examined at ages 7-8 he may have gradually "caught up" but now lag at the next hierarchical level of development (e.g., visual-motor integration).

Hypothesis 1. Younger dyslexic children (Ages 7-8) will be more delayed in visual-motor integration and auditory-visual integration than younger control children (Ages 7-8).

Hypothesis 2. Older dyslexic children (Ages 11-12) will not be more delayed in visual-motor integration and auditory-visual integration than older control children (Ages 11-12).

Hypothesis 3. Older dyslexic children will be more delayed in language integration skills than older control children.

Hypothesis 4. Younger dyslexic children will not be more delayed in language integration skills than younger control children.

Test of Hypotheses. The first direct test of the theory was carried out by Satz, Rardin and Ross (1971) on a small sample (N=40) of male children (dyslexic and control) at ages 7-8 (younger) and ages 11-12 (older). The children (Caucasian) were all sampled from a middle class elementary school; the experimental children (N=20) were initially identified by classroom teachers and were later screened to ensure that WISC Performance IQ was $\geq$ 90 and that reading performance was below grade level. The control children (N=20) were then selected to match for Performance IQ, sex, race

and age. The dependent variable measures included six tests, three of which were presumed to assess "earlier" developing skills (recognition-discrimination, perceptual-motor and auditory-visual discrimination) and three "later" developing skills (verbal intelligence, verbal fluency and ear asymmetry). The results showed that one of the earlier developing skills, Bender-Gestalt reproductions, were significantly delayed in the younger dyslexic group (Hypothesis 1) but that none of these measures differentiated groups in the older ages (Hypothesis 2). These findings can be visualized more readily by inspection of Figures 3 (Bender-Gestalt) and 4 (Auditory-Visual). With respect to Hypothesis 1 it can be seen that performance on the Auditory-Visual Test was also lower for the younger dyslexic than control group, but the difference was not significant.

Insert Figures 3 and 4 about here

By contrast, the results were more consistent for the "later" developing skills. Performance on each measure was significantly lower in the older dyslexic than control group (Hypothesis 3) but that these differences were not significant between the younger age groups (Hypothesis 4). These findings are presented in Figures 5 (VIQ - PIQ), 6 (verbal fluency) and 7 (ear asymmetry).

Insert Figures 5, 6 and 7 about here

Inspection of these figures reveals a dramatic change in the graphs, in comparison to Figures 3 and 4, with lower scores being observed in the older dyslexic children. For example, a minus discrepancy score between WISC VIQ and PIQ in Figure 5 reflects a depressed Verbal IQ which occurred only in the

older dyslexic group. Similarly, Figure 6 shows that the number of words produced to aurally-presented letters (F, A, S) was significantly lower only in the older dyslexic group. The younger children (dyslexic and control) produced similarly low percentile scores on the Verbal Fluency Test. The critical test of Hypotheses 3 and 4, however, rested on the results of the ear asymmetry data (Figure 7). The reason is that the lower scores on the language measures in the older dyslexic children could be explained on the basis of poor reading ability which over time would limit or retard related language skills (Belmont & Birch, 1966). Therefore, the dichotic listening test (Satz, 1968) was used to assess the magnitude of the ear asymmetry (i.e., cerebral speech dominance) between groups as a function of age. Figure 7 shows that the right ear asymmetry was developed in both groups as early as ages 7-8 which suggests that lateralization of the speech mechanisms already favored specialization on the left. The major test, however, was whether the degree of left hemisphere dominance for speech was delayed or less complete in the older dyslexic children. The results confirmed this hypothesis without showing any difference in total recall (R + L) between groups.

Thus, the results of this initial study lent substantial support for the theory, particularly for Hypotheses 2-4. Some of the measures of "early" developing skill differentiated groups in the younger children (Hypothesis 1) whereas none of these measures differentiated groups in the older children (Hypothesis 2). Conversely, all of the measures of "later" developing skill differentiated groups in the older children (Hypothesis 3) whereas none of these measures differentiated groups in the younger children (Hypothesis 4).

Additional, although less direct, support for the theory was reported in three recent studies. Kinsbourne (1971) investigated the perceptual

accuracy of two different age groups of retarded readers on a task involving rotated lamb chop designs. He found that the younger children (Ages 6-8) made significantly more errors on this task than did the older children (Ages 8 1/2-10). In fact, 85 percent of the younger group failed on one or more elements of the task (particularly orientation) in contrast to only five percent of older group. Thus, by ten years of age maturation had produced nearly errorless discrimination in the older retarded readers.

In a larger study, Sabatino and Hayden (1970) conducted a principal components analysis of several psycholinguistic and perceptual measures which were given to a younger (Age 7-7) and older (Age 11-4) group of disabled learners (N=472). They identified a primary loading on perceptual deficits in the younger children and a psycholinguistic deficit in the older children. Consistent with the developmental hypotheses already discussed, the authors concluded ". . . that six years to nine years is the maximum growth period for perceptual functional performance. After age ten, integrated language skills become of prime importance" (p. 411).

The findings of Kinsbourne (1971) and Sabatino and Hayden (1970) are in essential agreement with a study by Sparrow and Satz (1970) which examined the performance of an older group of disabled readers and matched controls (Ages 9-12) on a number of sensori-motor and cognitive-language tasks. Although younger age groups were not sampled, the study found group differences on only those tasks which assessed more complex cognitive and language skill. That is, the main effects were associated with those skills which are presumed to develop ontogenetically later. These results (Sparrow & Satz, 1970) are felt to lend additional support for Hypotheses 2 and 3 in the theory.

While each of the preceding studies provide at least indirect support for the theory, they can be criticized for a number of reasons. First, the

study by Kinsbourne (1971) merely focused on one specific measure (i.e., lamb chop designs) and used only disabled readers. Second, Sparrow and Satz (1970) failed to include younger dyslexic and control children although they utilized multiple measures of sensori-motor and cognitive skill. Third, the initial study by Satz et al. (1971), while designed as a direct test of the theory, was based upon limited N and restricted measures, at least for the "early" developing skills. Fourth, the study by Sabatino and Hayden (1970), while based on large sample size and multiple tests, was not designed as a test of the theory. Consequently, only indirect test of the hypotheses was possible.

In an effort to circumvent these and related criticisms, a larger and more direct test of the theory was undertaken by Van Nostrand (1972) in a recent doctoral dissertation. The primary effort was directed towards increasing sample size and the selection of tests for the "earlier" developing skills. An attempt was also made to control statistically for the concomitant effects of IQ and socio-economic level on the dependent variable scores. For this reason, a linear multivariate analysis of covariance was employed so that a separate analysis of covariance could be run on each of the adjusted measures, or a composite analysis of covariance could be run on all of the "earlier" developing skills or "later" developing skills. Twenty dyslexic children were selected at each of two different ages--younger (ages 7-8) and older (Ages 11-12)--and were matched with 40 control children on the basis of age, sex, race and school. Initial selection of Ss was again based on teacher recommendations and later screened to insure that WISC Performance IQ was $\geq$90.

Two major composite analyses of covariance were run. The first analysis was based on the "earlier" developing skills which included eight different measures of perceptual, perceptual-motor, auditory-visual,

right-left discrimination and finger tapping performance. Verbal IQ and socio-economic level were used as co-variates in this analysis. This composite analysis then permitted separate analyses of covariance for each of the eight measures by age (young vs. old) and by group (dyslexic vs. control). The second analysis was based on "later" developing skills which included three different measures of language performance (WISC Similarities, Verbal Fluency and Peabody IQ). Performance IQ and socio-economic level were used as co-variates in this analysis. This composite analysis then permitted separate analyses of covariance for each of the three measures by age and by group.

The results were again less consistent for the "earlier" developing skills (particularly Hypothesis 1) than for the "later" developing skills (Hypotheses 3 and 4). The composite multivariate analysis of covariance of the "earlier" developing skills revealed a significant main effect for ages ($F=16.13$, $p < .001$) and for groups ($F=2.28$, $p < .05$) although the age by group interaction was not significant ($F=1.36$, $p > .10$). When the separate analyses of covariance were run it was found that all of the measures revealed a significant improvement with age, but that only WISC Block Design performance differentiated between groups. Thus, performance on this measure accounted for the main effect for groups in the composite analysis. Figure 8 reveals the adjusted means for Block Design performance by age and by group.

Insert Figure 8 about here

Inspection of this figure shows that the predicted age by group interaction did not occur (Hypothesis 1) and that group differences were observed for both ages. It was only on the Alphabet (spoken and written) that a

significant age by group interaction occurred with lower performance evident in the younger dyslexic group (Hypothesis 1). This interaction can be seen in Figure 9. Figure 10 reveals a similar, though non-significant, trend for

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Insert Figures 9 and 10 about here
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performance on the Right-Left Discrimination Test. Thus, Hypothesis 1 was only minimally, if at all, supported by these analyses. Hypothesis 2, on the other hand, was substantially confirmed by virtue of the fact that only one of the eight measures differentiated groups in the older ages (i.e., Block Design).

The results of the second group of analyses (i.e., "later" developing skills) provided support for Hypotheses 3 and 4. The composite multivariate analysis of covariance revealed a significant main effect for ages ($F=18.97$, $p < .001$) and for ages by groups ($F=5.07$, $p < .01$). When the separate analyses of covariance were run, it was found that all three measures revealed a significant improvement with age which supports the selection of these tests as developmental measures. These developmental trends are easily visualized by inspection of Figures 11 and 12. The age by group interaction was largely

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Insert Figures 11 and 12 about here
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accounted for by performance on the WISC Similarities subtest (Figure 11) and the Peabody Test (Figure 12). In other words, performance on these tests was significantly lower in the older dyslexic group which lends support for Hypothesis 3. Again, there was no evidence of any difference on these language measures between the younger groups which provided further support for Hypothesis 4.

B. EVALUATION OF THEORY: CONCLUSION

The preceding findings, while somewhat inconsistent, were generally in support of the theory. Although some of the hypotheses still lack convincing support, particularly Hypothesis 1, the results substantiate the age variable as a relevant source of variation in developmental dyslexia. This variable has consistently been overlooked in previous studies which suggests that failure to isolate the effects of age could increase performance variability and consequently wash out meaningful differences either within dyslexic groups or between dyslexic and control groups. In fact, much of the controversy concerning the nature of this disorder could be explained on the basis of age variation effects. Instead of asking whether the primary handicap in these children is perceptual (Benton, 1962; Frostig, 1967; Ackerman, et al., 1971), linguistic (Benton, 1962; Belmont & Birch, 1966; Mason, 1967) or both (Ingram, 1970), the present findings suggest that the handicap will vary largely as a function of the chronological age of the child. Although there was less direct support for Hypothesis 1 which predicted that "earlier" developing skills would be more delayed in younger dyslexic groups, it should be noted that, in older age groups, no differences were observed

as a rule on these same measures (Hypothesis 2). Moreover, while most of the studies demonstrated lower performance in the older dyslexic groups on the "later" developing language skills (Hypothesis 3), no differences were observed as a rule in the younger groups on these same measures (Hypothesis 4).

It is interesting to note that in Benton's (1962) comprehensive review of the literature on the nature of the perceptual deficit in specific reading disability, he found that the problem was intrinsically age-related. Younger disabled readers showed a higher incidence of perceptual and visual-motor deficits than did older disabled readers. In fact, the perceptual problems tended to attenuate with age.[6] On the other hand, those studies which have challenged the perceptual deficit in disabled readers have generally sampled from older age groups (Ball & Owens, 1968).

The fact that the nature of the disorder varies, at least in part, as a function of chronological age suggests that a multiple etiology explanation need not be invoked to account for the variety of disturbances observed in these children (Ingram, 1970). The present theory at least provides a more parsimonious account of the behavioral variability in developmental dyslexia than speculation based on unobservable etiologies. Such speculation would tend to postpone or prevent attempts to uncover lawful regularities in their behavior. It was also shown that the age variable is a critical factor in both developmental psychology and neurology. Both disciplines recognize an increased maturation of the brain with age and a corresponding differentiation and growth of cognitive function. Moreover, this process is characterized by orderly and sequential stages, each of which depends upon the previous one for its development (Bruner, 1968; Hunt, 1961; Thurstone, 1955).

This concept of increased differentiation with age was observed in both the normal and dyslexic children who were reviewed in the previous section

(II-A). It was shown that the age or developmental differences were more striking, in fact, than the group or age by group differences. That is, substantial differences were uniformly observed between the younger and older age children on each measure, regardless of group membership. Although the results were based on cross-sectional rather than longitudinal designs, they suggest that the dyslexic child, even if delayed at Ages 7-8 on "earlier" developing skills (Hypothesis 1), tended to "catch up" on these skills by ages 11-12 (Hypothesis 2). This was not, however, the case for skills which are presumed to have a "later" or slower rate of development. Although no differences were generally observed between the younger age groups on these measures (Hypothesis 4), by Ages 11-12, substantial differences were evident (Hypothesis 3). These results therefore suggest that while the dyslexic child may eventually "catch up" on earlier developing skills or milestones, he may then lag in those skills which develop ontogenetically later. The fact that language-mediated skills have a slower or later rate of development (Thurstone, 1955; Bruner, 1968) would explain, in part, why the older dyslexic child was so consistently delayed on these tasks.[7] This delay in language-mediated skills at Ages 11-12 was not only consistent but quantitatively more severe than the perceptual lags that were occasionally observed in the younger dyslexic children. This finding parallels the observation of Lenneberg (1967) that the relative distances between developmental milestones for normal and retarded children become greater with advancing age (Figure 2). In the retarded child the earliest milestones were delayed by just a few months, but with advancing age the later developing milestones lagged even further behind the norm, even though the retarding disease was stationary and maturation progressed steadily but slowly. There is a marked similarity between Lenneberg's position and the findings relative to dyslexic children. The similarity refers to the observation that both the degree

and nature of the lag may change as a function of age with more severe delays associated with later developing skills. Intuitively, this interpretation is reasonable in that it postulates a lag mechanism which delays the acquisition of skills rather than a disease mechanism which produces a loss of corresponding skill. However, if the lag is later proved to increase with age or for later developing skills, then attempts will be necessary to determine whether the mechanism(s) underlying this maturation lag can be altered before the cognitive language disability is evident. The advantages of obtaining early indices of reading or perceptual handicap is that remedial programs may be introduced at a time when the child's CNS is more plastic and when psychological conflicts associated with reading disability are rare. There is some evidence to suggest that the child may be more sensitive to environmental stimulation (e.g., remedial intervention) during that period in which maturation of the brain is evolving and when behavior is less differentiated (Caldwell, 1968). Infrahuman studies (Scott, 1968) also suggest that organization can be strongly modified only when active processes of organization are underway and that when facilitated, they progressively inhibit attempts at reorganization. It has already been suggested that the remedial treatment of dyslexic children is more refractory when instituted after puberty (de Hirsch, et al, 1966).

The preceding conclusions, and the data from which they were extrapolated, have at least an initial "ring" of truth about them. Preliminary support was demonstrated for the hypotheses which were in turn based upon corroboratory evidence from the fields of developmental psychology and neurology. Support for the hypotheses, however, merely provided indirect confirmation for the mechanism postulated to underlie the disorder (i.e., lag in maturation of left hemisphere). Nevertheless, there is increasing evidence, based upon recent neurological and electrophysiological investigations, to suggest that

the underlying mechanism is somehow associated with cortical or left hemisphere asymmetry. First, the pattern of behavioral disturbances observed in the dyslexic child are frequently observed in adults who have sustained damage to the left hemisphere (Satz & Sparrow, 1970). Second, Luria's (1970) analysis of the reading and writing process (e.g., evaluation of speech sounds, word recognition, coding of sounds units, letter sequencing and language expression) showed that these components are selectively disturbed in adults with acquired focal lesions of the left hemisphere. Third, electrophysiological studies of dyslexic children have recently demonstrated either attenuation of the visual evoked response (VER) in the left parietal area (Conners, 1971) or less completely lateralized electrical activity (EEG) in the left hemisphere (Newton, 1968). Fourth, Childers, Ross, Perry and Nevis (1970) demonstrated a phase reversal in the VER in a group of familial and developmental dyslexics which further implicates an anomaly in cortical organization or assymmetry. Fifth, the reading process in later stages is intrinsically a language-mediated process (Gibson, 1968; Luria, 1970) which is facilitated by the left cerebral hemisphere in right-handers and the majority of left-handers (Satz, Achenbach & Fennell, 1967). In fact, Benton (1962) earlier concluded:

> Impairment in oral and silent reading is a common feature of aphasic disorders. In most cases, it is reasonable to think of the observed reading disability as being one further aspect of a total syndrome of language disturbance which is reflected in all modes of comprehension and expression of symbolic material (p. 18).

Thus, while the evidence points consistently to some type of underlying alteration in left hemisphere organization, the mechanism remains obscure. The theory advanced in this chapter (Satz & Sparrow, 1970)

postulates that a lag mechanism may be involved, primarily delaying the maturation of the left cerebral cortex. It was earlier shown that the concept of a maturational lag has already been advanced as a possible factor in developmental dyslexia (de Hirsch, et al., 1966; Money, 1962). These authors have also maintained that maturation of the CNS is largely a function of chronological age. Evidence was presented in the preceding section which showed that parameters of brain maturation and cognitive organization increased as a function of age. The theory predicts that a lag in the maturation process will retard the corresponding acquisition of behavioral skills. Thus, the developmental level of older dyslexic children should resemble the performance of chronologically younger normal children.[8]

This formulation conveniently avoids the use of terms, usually pernicious, which conceptualize the disorder within the framework of a static disease model (e.g., brain damage). The evidence strongly suggests that these children, regardless of etiology, are not fixated in development and that behavioral milestones are attained if only at a slower rate. Unfortunately, this observation has all too frequently been overlooked in studies of the handicapped learner.

De Hirsch and associates (1966) have demonstrated the most convincing support for a maturational lag hypothesis in dyslexia. This was accomplished by obtaining multiple measurements on a small group of pre-school children and following them through the second grade at which time reading achievement was assessed. The thirty-seven tests were divided into those which were maturation-sensitive and those which were not. It was shown that later reading achievement was largely predicted by the maturation-sensitive tests (76 vs. 17 percent).

Although the preceding evidence lends considerable weight to the formulations concerning a possible lag in the maturation of the left

hemisphere, the nature of the underlying mechanism still remains obscure. Microscopic studies of brain maturation have not yet been conducted in these children and evidence based on infrahumans has been shown to be exceedingly complex (Pupura, Shofer, Housepian & Noback, 1964). Thus, the present formulation must remain at the level of constructs hypothesized to account for unobservable events.

An additional and not unrelated problem concerns the possible role of genetic mechanisms in developmental dyslexia. Silver (1971) has recently presented data which points to a striking familial pattern in approximately forty percent of children with specific reading disability. While it is unclear as to whether these children meet the criteria for developmental dyslexia, the results could be interpreted within the context of the theory already advanced (Satz & Sparrow, 1970). That is, it is possible that the underlying mechanism, genetic or otherwise, could result in a pre-disposition to immature patterning (maturation lag) which in turn retards the acquisition of reading and related developmental skills. Clarification of this problem, however, will have to await developments in both neuropathology and behavioral genetics. It is interesting to note, nevertheless, that other sources of data point to a sex-linked or constitutional factor in dyslexia. Studies have uniformly reported a disproportionately higher incidence of males in this disorder (Eisenberg, 1966; Satz & Sparrow, 1970). In the context of the present theory, developmental and neurological studies have long shown that boys maturate at a slower rate than girls, particularly during the first seven years. This sex-lag phenomena has been especially true for early language and perceptual development (Beery, 1967; Kimura, 1967; Sabir, 1966). These findings consequently shed further indirect support to the hypothesis that many of the high risk children who enter

pre-school, particularly boys, may not be maturationally or developmentally "ready" to cope with the early formal demands of reading.[9] This possibility again underscores the need to develop early and valid predictors of later reading disability which will foster intervention at a time which may be more optimal for the child and the schools.

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FOOTNOTES

1
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2
The authors gratefully acknowledge the continued help and permission which was granted by Ms. Christine Clark, Principal of Sidney Lanier Elementary School, Gainesville, Florida, and Ms. Janette Friel and the staff of the Neuropsychology Laboratory who labored in collecting the data.

3
This hypothesis is based on the observations of Bloom (1964) that variations in the environment have their greatest quantitative effect on a characteristic at its most rapid period of change and the least effect at the least rapid period of change. It is assumed that such effects would be equally triggered by variations in CNS maturation.

4
It is recognized that the order or sequence of developmental stages is more orderly than the specific age at which each stage appears (Hunt, 1961).

5
Although Lenneberg focused on the behavioral effects of brain maturation in mental retardation, his formulation could be extended to other clinical phenomena, such as dyslexia, particularly if one postulates a quantitative rather than qualitative difference between conditions.

6
This age factor was recently shown to affect the relationship between tests of motor skill and the criterion of academic achievement in normal elementary school boys (Chissom, 1971). A relationship between motor skills (balance and motor coordination) and academic achievement or aptitude was demonstrated in first grade boys but not in

third grade boys. The results suggest that skills which undergo development ontogenetically earlier are more likely to be correlated with earlier criterion measures of academic achievement and aptitude. With respect to intervention programs, the results further suggest that perceptual-motor training programs (e.g., Kephart, 1960) may be more applicable or effective when administered to younger learning disabled children.

7 This interpretation, however, should be tempered by the fact that different children were sampled at each age group. Again, this points out the need for longitudinal research in which observations can be made on the same child at different ages. The senior author is presently engaged in a large-scale longitudinal study of all male kindergarten children (Caucasian) in Alachua County, Florida.

8 In this context, chronological age should not provide a reliable index of developmental level in dyslexic children due to an underlying lag in neurophysiological maturation.

9 The lag mechanism still presents difficulties in explaining why some children who are delayed in pre-school eventually "catch up" without developing reading problems (de Hirsch, et al., 1966) and why others, while developing at a slower rate, show persistent and longterm reading disability effects. Although genetic mechanisms may be involved in both cases, it does not rule out the possibility that failure to overcome the handicap before maturation of the CNS is complete could lead to more persistent, if not irreversible, effects. Lenneberg's data (Figure 2), and the data reviewed in Figures 3-12, strongly suggest that the behavioral disturbance becomes progressively more severe as the child becomes older. This evidence, however, is not based on

those children (e.g., slow starters) who may have eventually caught up and overcome their handicaps. This problem will require more careful and systematic longitudinal investigation.

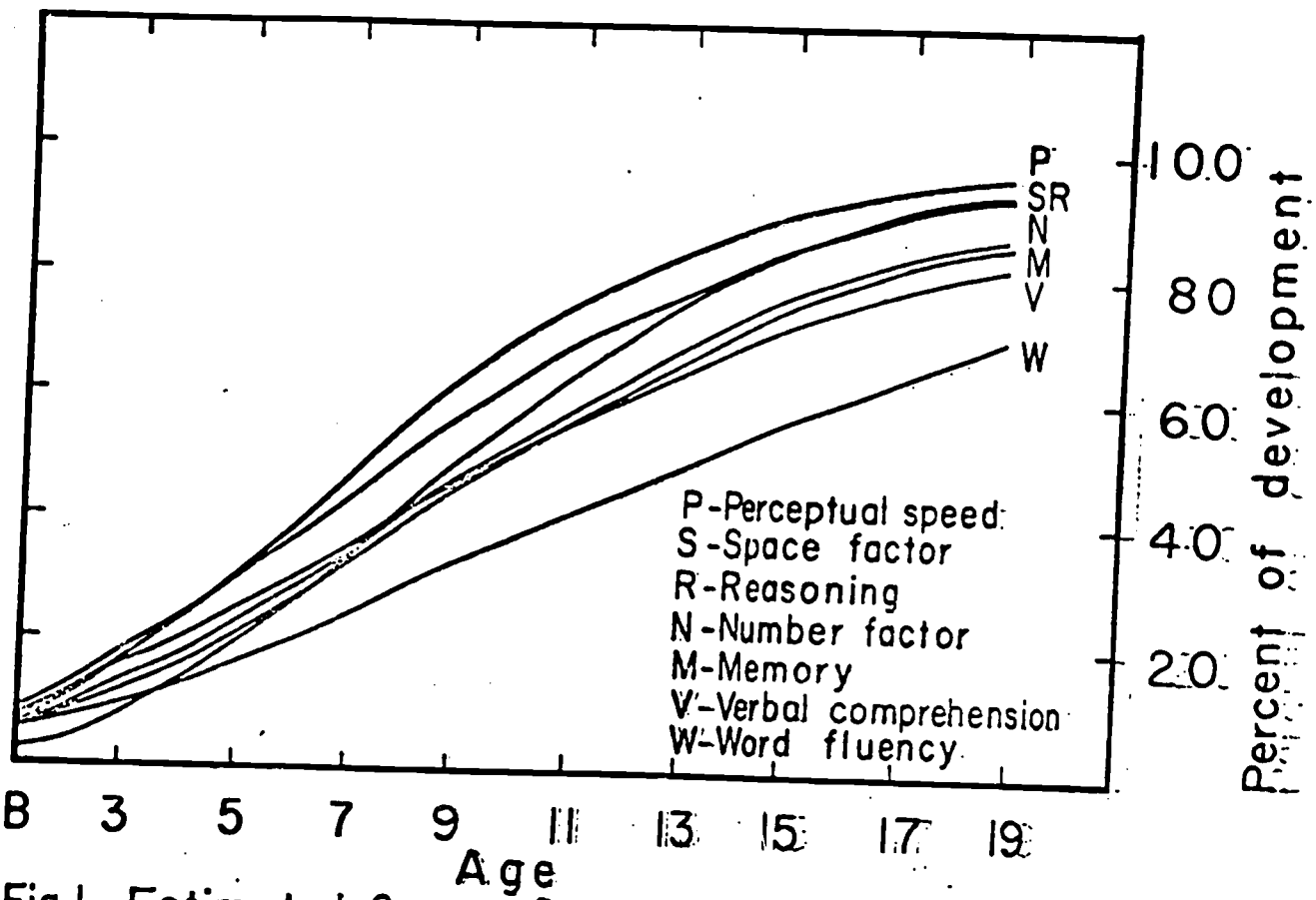

Fig.1. Estimated Curves for the Development of Specific Mental Abilities (Thurstone, 1955).

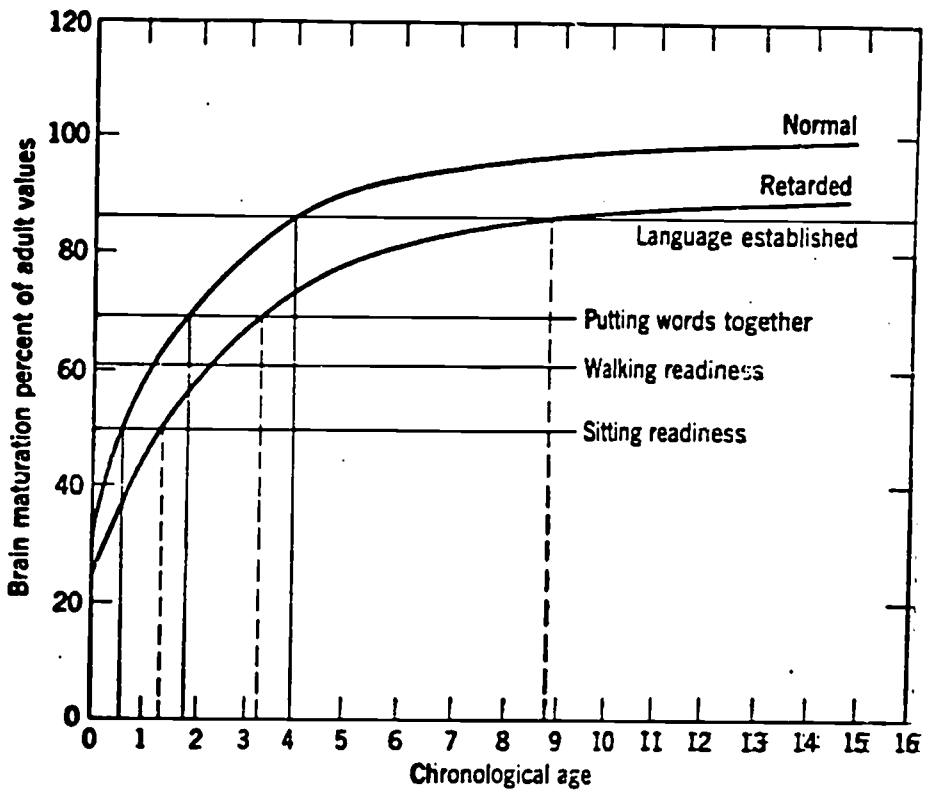

Fig. 2. Brain maturation and developmental "horizons" by age for normal and retarded children (Lenneberg, 1967).

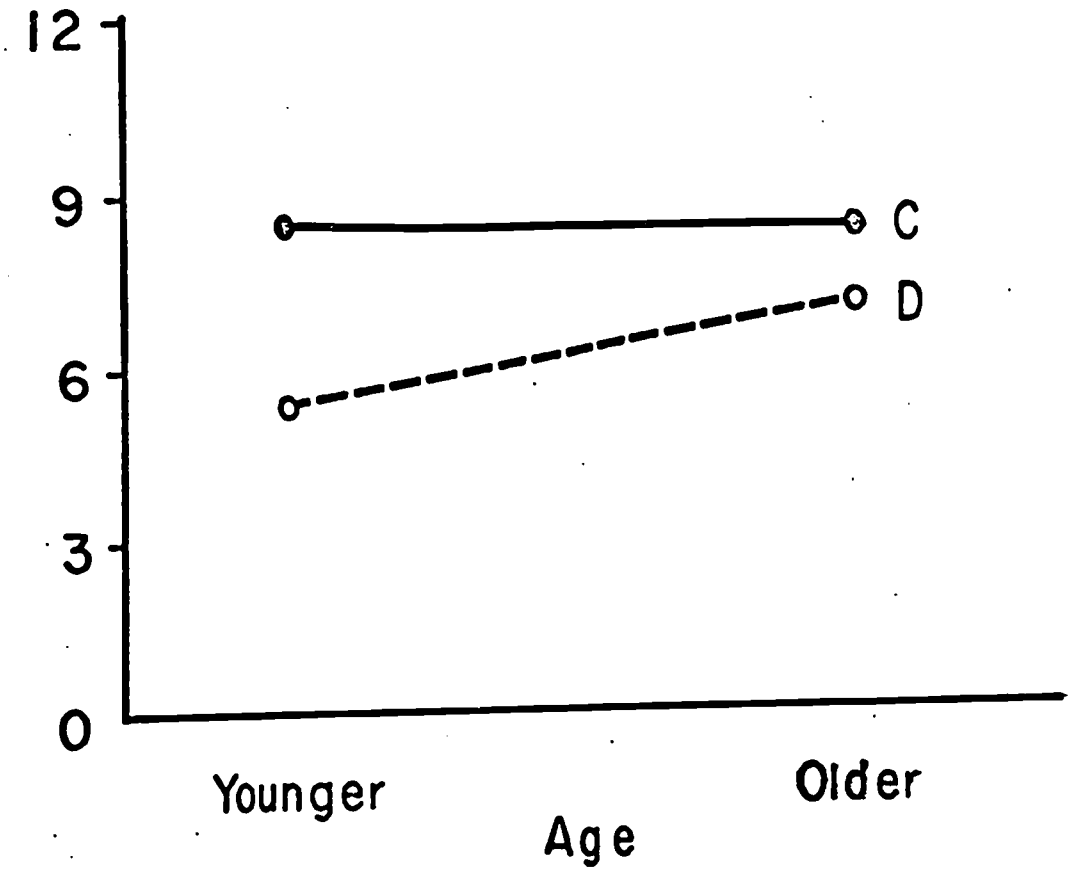

Fig.3. Bender-Gestalt scores by age and group.

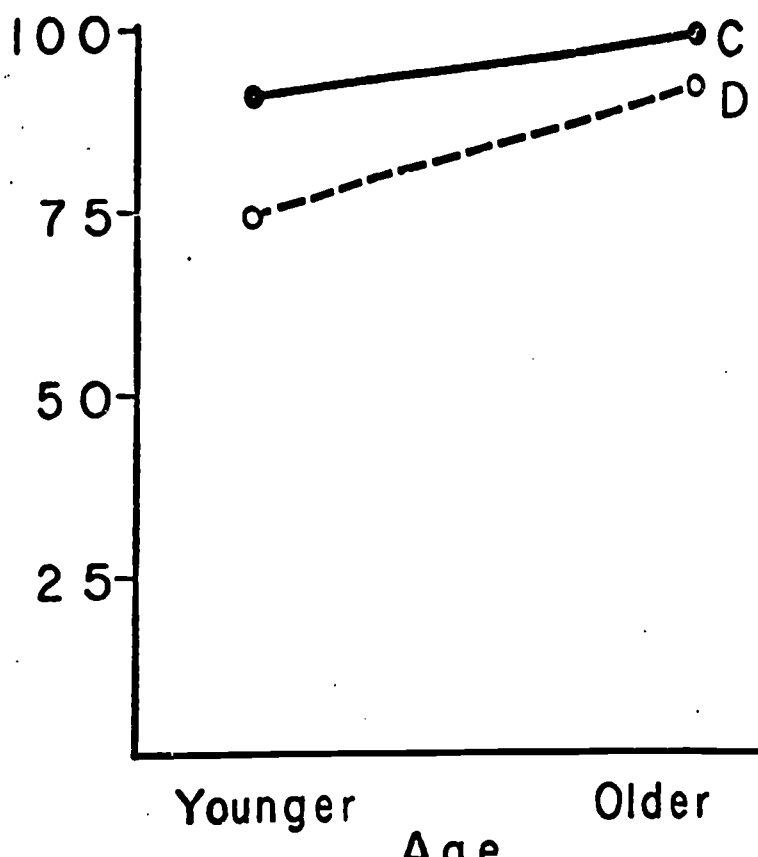

Fig. 4. Mean Auditory-Visual scores (% correct) by age and group.

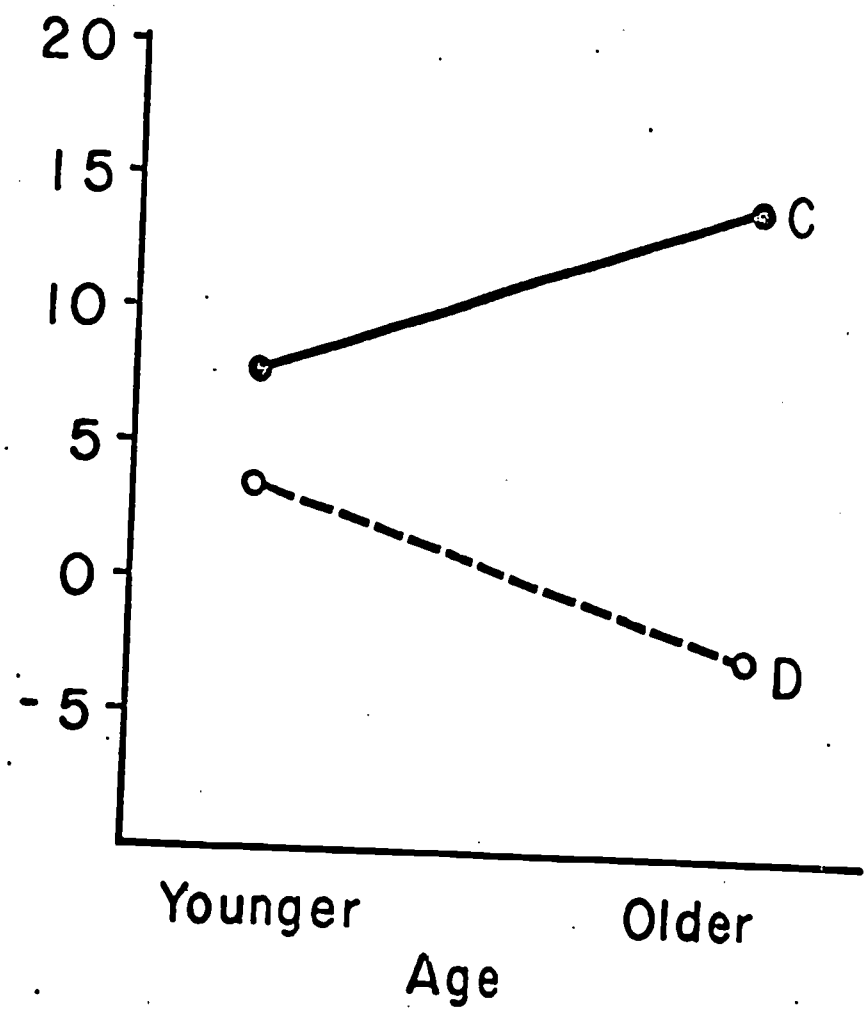

Fig. 5. Mean VIQ-PIQ difference scores by age and group.

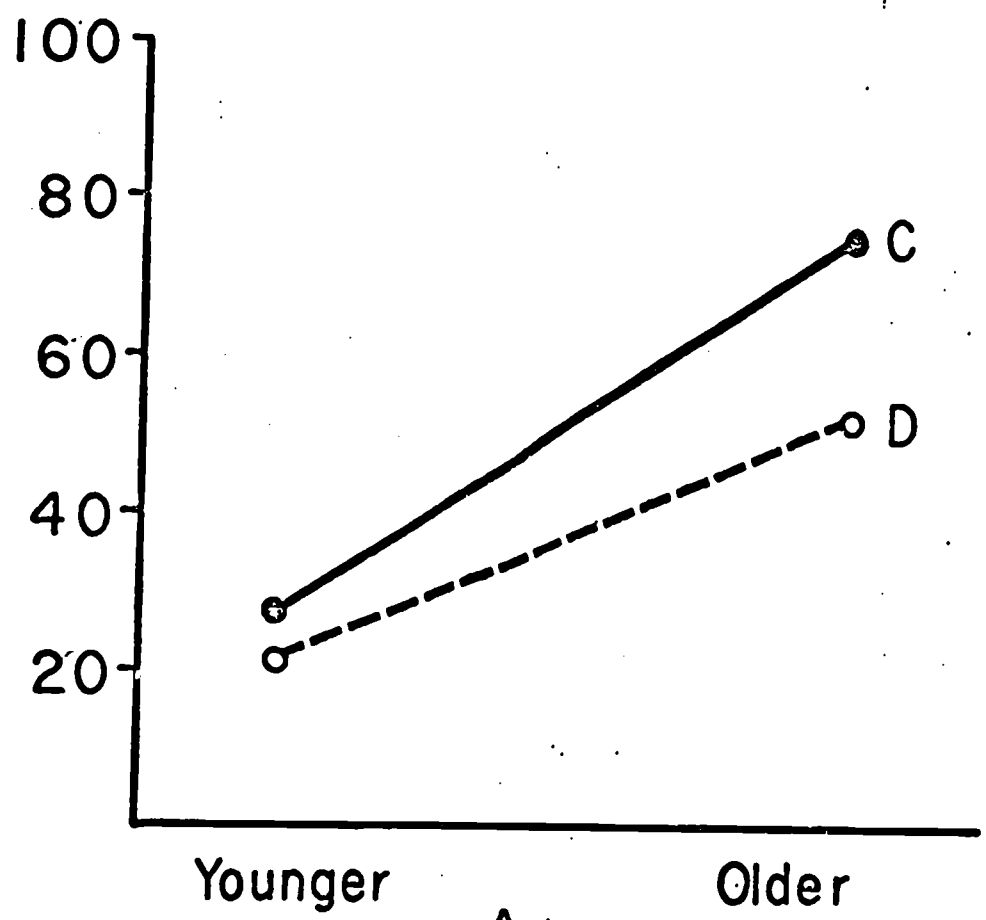

Fig.6. Verbal Fluency scores (percentile) by age and group.

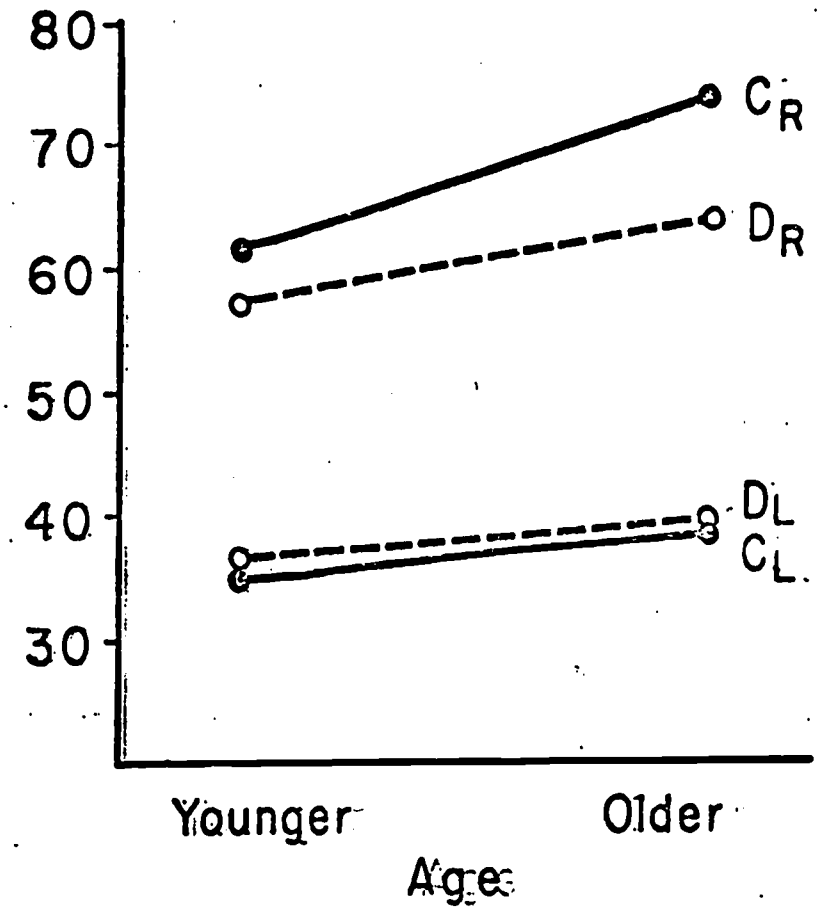

Fig. 7. Dichotic Listening scores (% correct, R+L) by age and group.

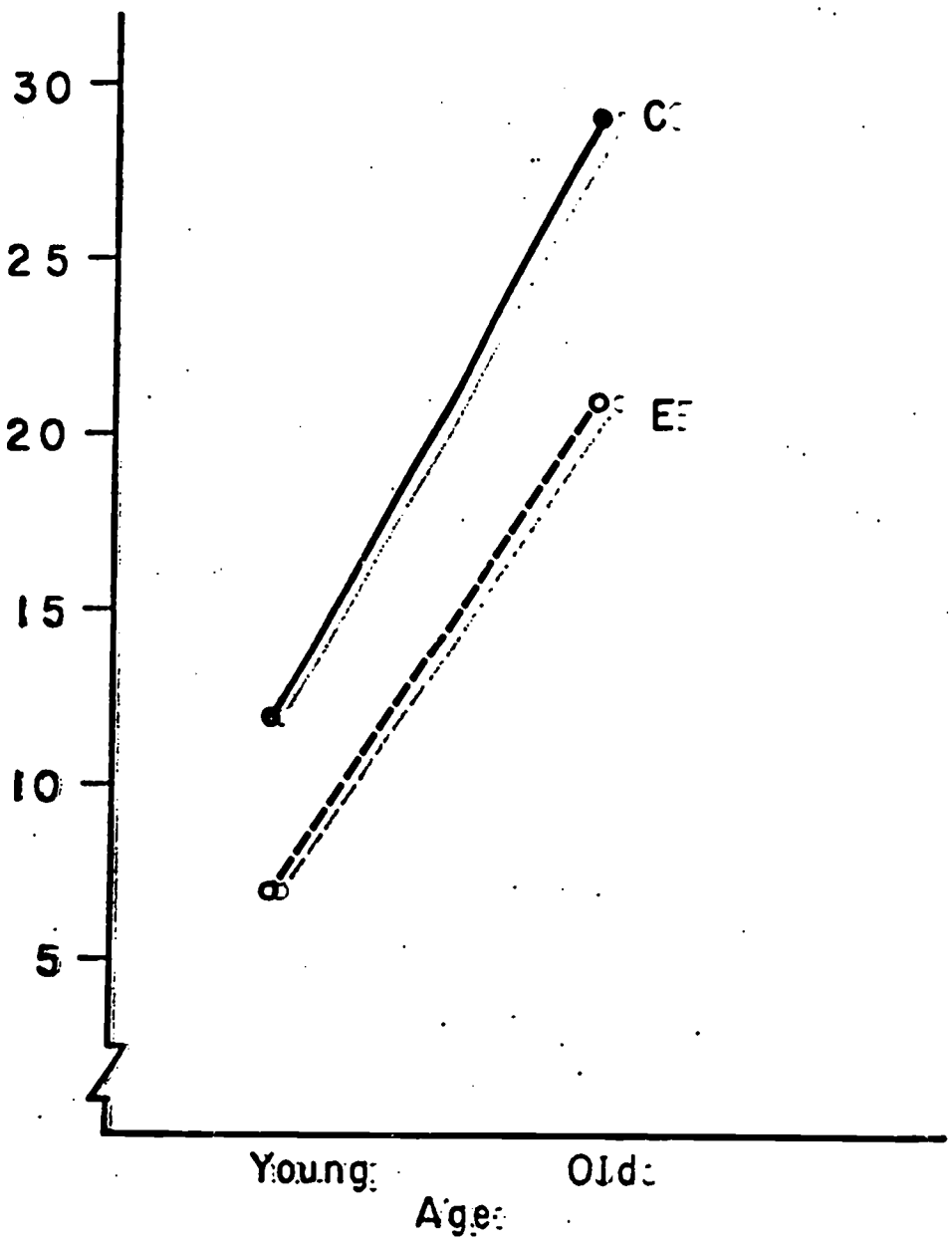

Fig.8.. Adjusted Means for Block Design (Raw Score) by age and group.

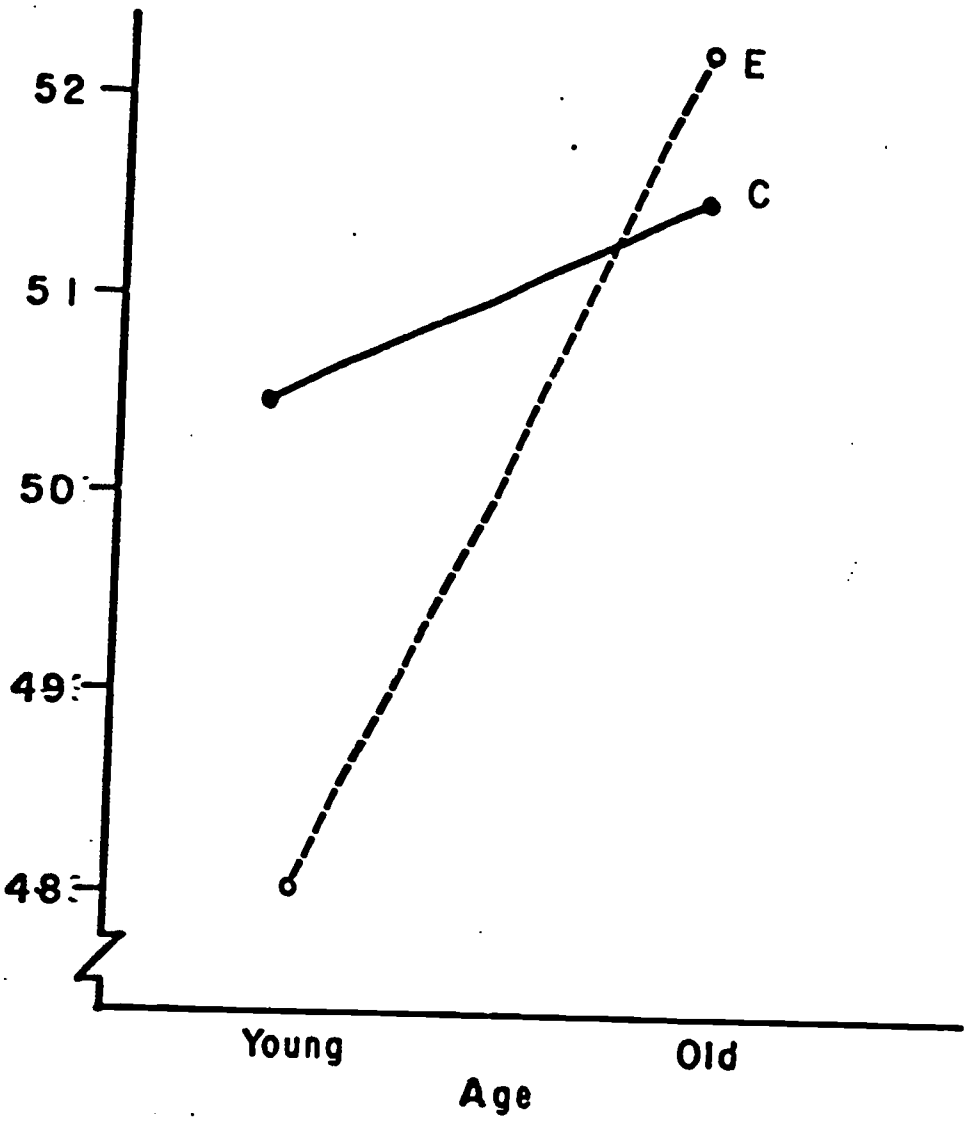

Fig. 9. Adjusted Means for alphabet (S + W) by age and group.

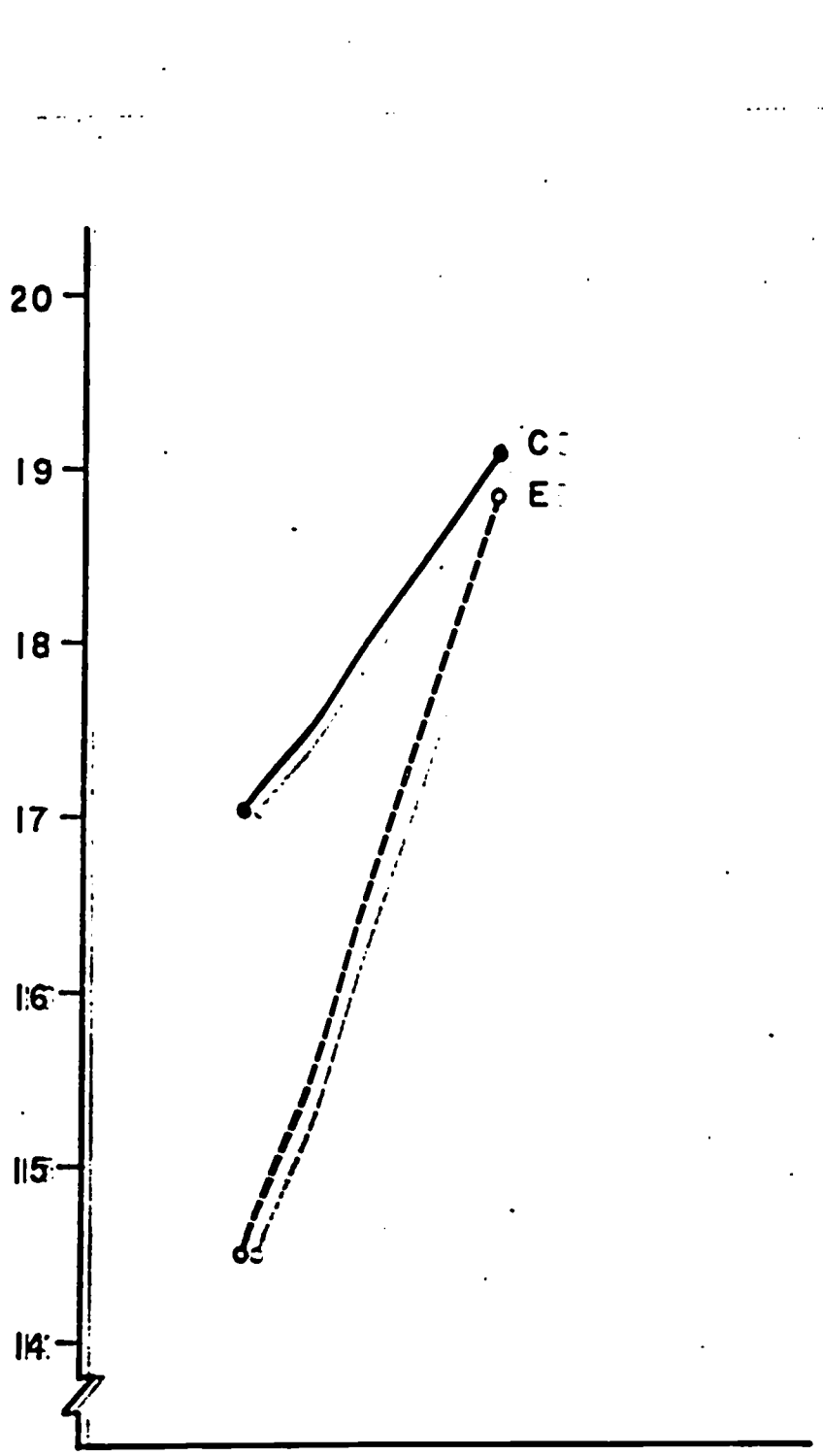

Fig. 10. Adjusted Means for Right-Left (A+B) by age and group.

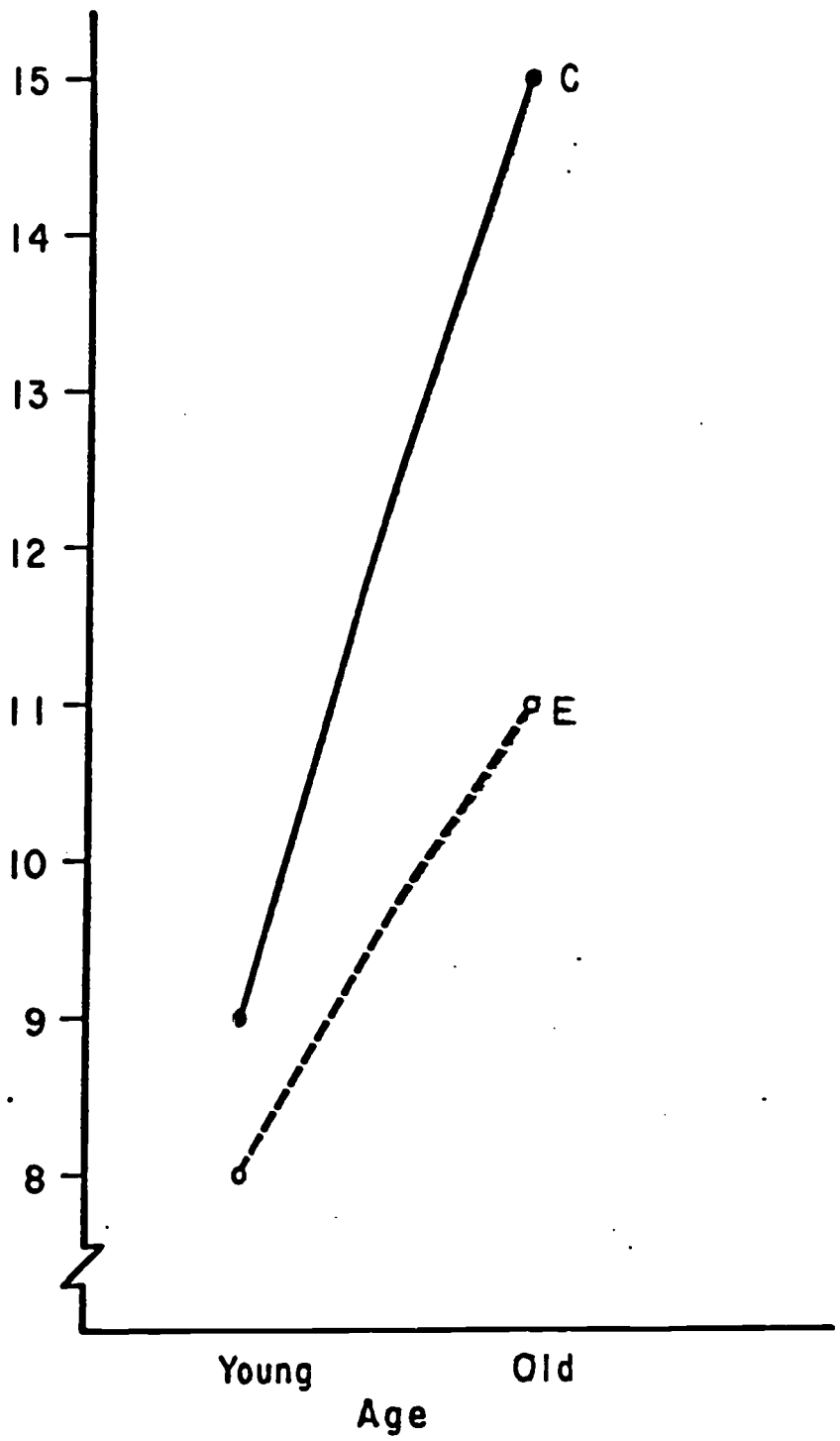

Fig. II. Adjusted Means for Similorities (Raw Score) by age and group.

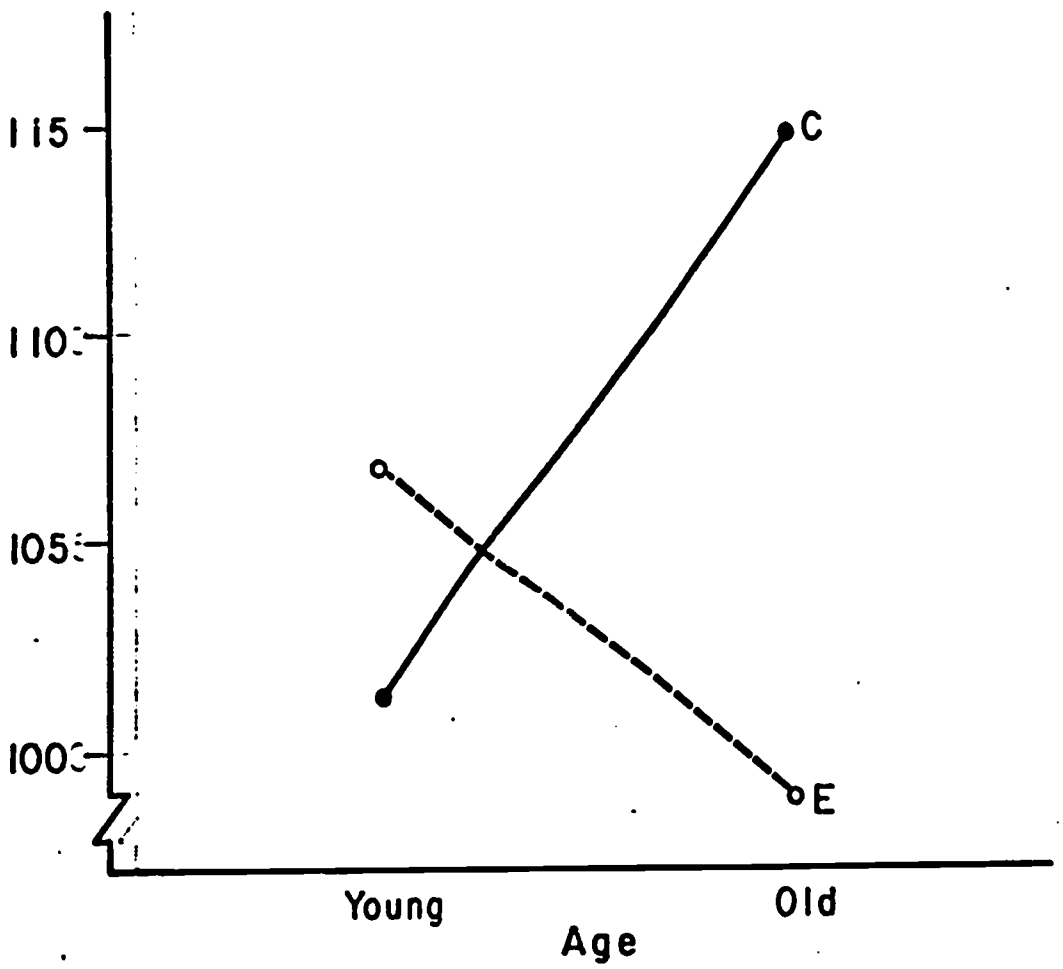

Fig. 12. Adjusted Means for Peabody IQ by age and group.